Contemporary mediastinal tuberculosis

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ABSTRACT Mediastinal lymphadenopathy is a prominent feature of adult tuberculosis in immigrant groups in the United Kingdom. Chest radiography of 95 tuberculous immigrants showed mediastinal gland enlargement in 29 cases, whereas none of the 42 Europeans showed this feature. An analysis has been made of the distribution of the disease in all groups together with a more detailed evaluation of mediastinal involvement in the immigrants. Pericardial effusions were seen in eight patients together with the following three unusual complications: (1) broncho-oesophageal fistula; (2) bronchial erosion; (3) superior mediastinal obstruction.

Mediastinal lymph node involvement has been an uncommon feature of intrathoracic tuberculosis in adults in the United Kingdom, whereas it is the rule in primary tuberculosis, especially in children. It is, however, frequently seen in adult immigrants of all ages, and extrathoracic disease is common.1 The epidemiological aspects of contemporary tuberculosis have been well recorded.2 3 The radiological aspects have received less attention.

Methods

The radiographs and case notes of all new patients presenting with tuberculosis at St James' Hospital in 1976 and 1977 were reviewed by one of us (CJD). A detailed analysis was made of the 137 patients who were aged 16 years and over. The racial origin was known—79 were Asians from the Indian subcontinent or East Africa, 42 were Europeans, and 13 were Blacks from Africa and the West Indies. In addition, two patients were Chinese and one was an Egyptian. Most were local residents, but a few were visiting the area when they became ill. The diagnosis was made in 95 out of 137 cases by isolation of the organism or by the presence of caseating granulomata. In the remaining patients, particularly those with mediastinal involvement alone the Mantoux reaction and response to treatment were the sole evidence of infection.

Results

The distribution of the disease is shown in table 1 and the pattern of mediastinal involvement in table 2. Although right-sided nodes can be differentiated into paratracheal and hilar, those on the left are difficult to separate and have been combined.

Thirty-seven of the 42 European patients showed lung disease (88%). Mediastinal nodes were not affected and the disease was present at a single site in all cases. The mean age was

<table>
<thead>
<tr>
<th>Site</th>
<th>European 42 cases</th>
<th>Non-European 95 cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lung (including pleural effusion)</td>
<td>37</td>
<td>37</td>
</tr>
<tr>
<td>Mediastinal nodes</td>
<td>0</td>
<td>29</td>
</tr>
<tr>
<td>Cervical nodes</td>
<td>2</td>
<td>28</td>
</tr>
<tr>
<td>Nodes elsewhere</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Pericardial effusion</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>Peritonitis</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Bone</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>Soft tissue abscess</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Ano-rectal</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Genital</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>CNS/renal</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Table 2 Sites of enlarged mediastinal nodes

<table>
<thead>
<tr>
<th>Site</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right paratracheal alone</td>
<td>12</td>
</tr>
<tr>
<td>Right hilar alone</td>
<td>5</td>
</tr>
<tr>
<td>Right paratracheal and right hilar</td>
<td>4</td>
</tr>
<tr>
<td>Right paratracheal and left hilar</td>
<td>3</td>
</tr>
<tr>
<td>Left hilar alone</td>
<td>5</td>
</tr>
<tr>
<td>Total</td>
<td>29</td>
</tr>
</tbody>
</table>

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Table 3 Mediastinal complications

<table>
<thead>
<tr>
<th>Complication</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pericardial effusion</td>
<td>8</td>
</tr>
<tr>
<td>Broncho-oesophageal fistula</td>
<td>1</td>
</tr>
<tr>
<td>Bronchial erosion</td>
<td>1</td>
</tr>
<tr>
<td>Superior mediastinal obstruction</td>
<td>1</td>
</tr>
</tbody>
</table>

50 years and women were in the minority (11:31).
In contrast 37 of the 95 non-Europeans showed lung disease (40%). Mediastinal glands were present in 29 cases (30%) but only 11 of these were associated with lung disease. In 15 cases these nodes were the sole manifestation of the infection. The mean age was lower (32 years). Women were in the majority in the Asians

Fig 1 Barium introduced via a tube in the lower oesophagus outlines the fistula between the oesophagus and the right main bronchus.

Fig 2 Atelectasis of the medial basal segment of the right lower lobe, caused by erosion of a tuberculous sub-carinal node into the right lower lobe bronchus.

Fig 3 Pericardial effusion and widened superior mediastinum.
(47: 32) but not in the Blacks (2: 13). The pattern of the disease was similar in Asians and Blacks. Both Chinese patients showed mediastinal node enlargement alone.

Complications in the mediastinum were seen in one African and nine Asian patients and details are shown in table 3. The tracheo-oesophageal fistula (fig 1) presented in a woman of 40 years with a normal chest radiograph. It closed after five weeks of chemotherapy. The bronchial erosion (fig 2) was seen in a youth of 16 years who had already been treated for three months for a cervical node infection. His tubercle bacillus was later shown to be insensitive to two of the original drugs. Finally, a man of 40 years with a pericardial effusion and right paratracheal node enlargement (fig 3) developed superior mediastinal obstruction (fig 4). He had already received three months adequate treatment. The radiological changes persisted six months later.

Discussion

Since Silver and Steel described seven adults with mediastinal tuberculosis in London in 1961, there have been reports of similar cases from many parts of the world. The patients described in 1961 were either West Indian or Asian immigrants, but it has been known for many years that Europeans could sometimes show enlarged intrathoracic glands and the subject was discussed by Pagel in 1942. Hess and MacDonald found a particularly high incidence of primary tuberculosis in the adult Irish population in London in 1954. The reasons why populations respond differently to tuberculosis has been reviewed elsewhere. It appears that environmental and racial characteristics play a part as well as previous exposure to infection. The tubercle bacillus is known to show a difference in bacteriophage-type pattern in African, British, and Asian groups. This does not appear to influence the site of adult infection or to account for the increase in extrapulmonary and nodal tuberculosis seen in immigrants in this country. Difference in susceptibility and immune reaction may be important factors. There are few comparable series from Asia, Africa, or the West Indies but in a study of tuberculosis in Nigeria in 1975, Kolawole et al found a pattern of mediastinal disease in children and in adults similar to that found in Europeans. In a review of 216 patients with tuberculosis of all ages, enlarged intrathoracic glands were found in 18 out of 50 of those under the age of 10 years but in only six out of 166 over the age of 10 years. Although this differs from our findings, the reason may be that patients in Nigeria present when their disease is considerably more advanced and the mediastinal lymphadenopathy is hidden by the parenchymal disease. Certainly our patients with mediastinal disease alone had only minor physical complaints. The difference in the behaviour of the disease in the populations studied is important to the radiologist, for there are many reports of cases originally misdiagnosed, usually as malignant disease. The site of enlargement agrees with the work of Amorosa —that is, that the right paratracheal nodes
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are those most commonly enlarged. The limitations of a simple chest radiograph in this respect are obvious as many nodes will be hidden. Right paratracheal with left hilar glands were seen in three patients. There were no examples of bilateral hilar lymphadenopathy but others have described this finding in tuberculosis.13–15 It has been suggested16 that in an asymptomatic patient, or in a patient with erythema nodosum or uveitis, bilateral hilar lymphadenopathy is diagnostic of sarcoid and further investigation is unwarranted. Our study does not refute this but the well-documented cases of others make it clear that tuberculosis cannot be ignored in the differential diagnosis of hilar lymphadenopathy in immigrants.

Fistulae between the oesophagus and respiratory tract have long been recognised in children with tuberculosis. Riviere in 190317 described the necropsy findings in three children with tuberculous fistulae, but the rarity of the condition in adults reflects the infrequency of mediastinal disease. Adenis et al18 described a tracheo-oesophageal fistula in a man of 71 years with enlarged posterior mediastinal lymph nodes, and two cases were described by Pecora.19 One of the patients had tuberculous disease of the thoracic spine before the fistula developed and in this case it was suspected that the disease had spread from the spine to the oesophagus and trachea. The case reported by Wigley et al20 is of particular interest for two reasons. First, the fistulous communication was between the right main bronchus and the oesophagus (as in the case shown in this paper), and second, the fistula closed on chemotherapy alone. They regarded theirs as the first reported example of a tuberculous fistula from the respiratory tract to the oesophagus that had closed without recourse to surgery.

Collapse of the lung in childhood tuberculosis is the result of compression of a bronchus by enlarged nodes or by erosion of caseating nodes causing endobronchial occlusion.21 In the absence of upper zone disease, collapse, or consolidation in the lower lung fields may have the same aetiology in adult immigrants. Berger and Granada22 described 27 cases and found hilar nodes frequently involved in lower zone disease. They considered that transbronchial perforation of the node caused the parenchymal lesion in many cases.

The most dramatic effects of lymph node enlargement in the superior mediastinum are again seen in infancy and childhood. As well as compression of the major veins, life-threatening compression of the trachea has been described.23 The trachea is less easily occluded in adults and mediastinal venous structures are the most vulnerable to compression. Malignancy is the common cause of the syndrome but granulomatous disease can occasionally be responsible. In the North American literature it is usually attributed to histoplasmosis rather than tuberculosis unless there is positive bacteriological evidence of tuberculosis. In the United Kingdom histoplasmosis is rarely seen and tuberculosis is the main cause. Three patients with superior mediastinal obstruction resulting from tuberculosis were described by Steinberg in 1966.24 In each case the cause was different. The first patient had chronic fibrosing mediastinitis. The superior vena cava of the second patient was compressed by caseous mediastinal nodes and in the third case calcified nodes in the superior mediastinum occluded the superior vena cava. Either of the first two mechanisms could have operated in our case. Not only the venous side of the cardiovascular system is susceptible to tuberculosis. We encountered eight examples of tuberculous pericarditis with effusion. Others have described a pericardial tuberculoma masquerading on the chest radiograph as an enlarged left atrium,25 and in a pathological study Behr et al26 described two cases of myocardial tuberculosis. Aortic involvement leading to myotic aneurysm formation has also occasionally been reported.26 27

In summary, this study has demonstrated the different patterns of tuberculous disease and the mediastinal pathology that can be recognised radiologically. Mediastinal lymph node enlargement caused by tuberculosis must be distinguished from the lymph node enlargement seen in sarcoid and malignant disease, and although the ethnic group has a strong discriminative value, a high index of suspicion is needed to diagnose atypical cases.

We wish to express our thanks to Dr FJC Millard for allowing us to publish this report on his patients, and also to Dr JC Batten for his advice in the presentation of the article. Our thanks are also due to Mrs W Aarons for typing the manuscript and to Mrs H Townsend of the Department of Thoracic Medicine, St James' Hospital for her help.

References

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